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The Clinical Features of Covert Diuretic Use

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DIURETIC AGENTS are prescribed for the treatment of hypertension, congestive heart failure, hypercalciuria and edema. The side effects of these drugs are easily recognized when their use is known. However, covert diuretic ingestion can mimic a variety of disease states and patients may endure long, expensive and painful investigations rather than admit to medicine abuse.

We recently saw two patients who were surreptitiously taking diuretics. Their clinical features had led to previous investigations for hyperparathyroidism, sarcoidosis, cholelithiasis, Sjögren's syndrome, pancreatitis, renal insufficiency and Bartter's syndrome.[1] Workups had been inconclusive until laboratory test findings disclosed the presence of furosemide or thiazide, or both, in the urine of both patients. Despite denials of diuretic use, these drugs seemed to play a major role in the development of these patients' disease states. The cases are presented to alert physicians to the wide spectrum of presentations possible in patients abusing diuretics.

Reports of Cases

CASE 1. A 29-year-old Native American woman was referred in July 1980 for evaluation of hypercalcemia and repeated bouts of pancreatitis.

In 1975 she had weighed 84 kg (185 lb). She lost 41 kg (90 lb) during the next three years with the use of diet and thiazide diuretics. She said she had not taken thiazides since June 1978. In April 1979 she complained of a dry mouth and arthralgias and underwent an evaluation for Sjögren's syndrome at a university hospital. The results of this evaluation were negative. Beginning in June 1979 repeated bouts of pancreatitis led to a gastrointestinal evaluation. Despite extensive investigations, including oral cholecystography and endoscopic retrograde cholangiopancreatography (ERCP), no cause for the pancreatitis was established. With the known high incidence of cholelithiasis in Native Americans, however, her repeated episodes of pancreatitis led to a cholecystectomy in August 1979. A normal gallbladder was found.

In September 1979, with a history of mildly elevated calcium and blood urea nitrogen (BUN) levels, she was examined for sarcoidosis and renal disease, with negative findings. In July 1980, due to mild hypercalcemia and persistent bouts of pancreatitis, she was referred to the endocrine

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clinic for evaluation of possible hyperparathyroidism.

She was a thin, anxious young woman who had symptoms of thirst, nocturia, abdominal discomfort and malaise. She said she did not have diarrhea or vomiting or use diuretics or laxatives. She noted that hydrochlorothiazide had been prescribed for her mother for hypertension. Findings on physical examination were unremarkable except for mild orthostatic hypotension.

Laboratory studies disclosed the following values: serum sodium 115 (normal, 135 to 145), serum potassium 3.4 (3.5 to 5), serum chloride 73 (96 to 106) and serum bicarbonate 20 mEq per liter (24 to 32); BUN 90 (8 to 24), creatinine 2.2 (0.6 to 1.4) and calcium 10.5 mg per dl (8.5 to 10.5); albumin 5.1 grams per dl (3.5 to 5), and uric acid 11.1 (3.5 to 8) and glucose 106 mg per dl. Results of liver function tests were within normal limits. Another chemistry panel gave the following findings: sodium 122 and potassium 2.9 mEq per liter, BUN 57, creatinine 1.7 and calcium 10.6 mg per dl.

Hypoadrenalism was considered, but ruled out by normal results on a one-hour cosyntropin stimulation test, with a peak cortisol level of 45 μg per dl. The hypercalcemia was thought to be due to dehydration, which was confirmed by repeatedly normal ionized calcium determinations. The serum parathyroid hormone concentration (Upjohn Laboratories, Kalamazoo, Michigan) was appropriate for the level of ionized calcium. Urinary calcium was 46 mg per gram of creatinine (normal, 50 to 250). Spot urine sodium was 1 mEq per liter and urine potassium was 10 mEq per liter. A serum aldosterone determination was done on a specimen taken while the patient was in a supine position and gave a value of greater than 300 ng per dl (normal, 2 to 9); plasma renin activity was reported 22.8 ng per ml an hour (normal, 1.1 to 7.9).

The electrolyte acid-base findings suggested a gastrointestinal-caused electrolyte loss. However, given the history of polyuria, xerostomia, hypercalcemia and pancreatitis, and the known maternal consumption of diuretics, we screened a urine specimen for thiazides. A urinary thiazide concentration of 7 μg per ml was found. Urine electrolyte studies done at the same time showed a sodium concentration of 2 mEq per liter and potassium of 27 mEq per liter; chloride was not measured. The low urinary sodium level in the presence of diuretic use suggested that gastrointestinal electrolyte loss was mainly responsible for her serum electrolyte abnormalities.

She subsequently admitted to having moderate diarrhea but she would not elaborate as to quantity or type of stool excreted (watery versus formed, and so forth). After confrontation, she continued to deny diuretic and laxative use. The patient was referred back to her primary physician with diagnoses of (1) hypercalcemia, xerostomia and pancreatitis due to thiazide abuse and (2) electrolyte abnormalities due to a combination of gastrointestinal and thiazide-induced losses.

CASE 2. A 30-year-old woman was referred to our endocrine clinic for evaluation of hypokalemia. She noted a purposeful weight loss from ages 12 to 20, and a psychiatric admission during adolescence. She gave a history of cyclic edema in her early 20's.

Six years previously she had been admitted to a university hospital for investigation of explosive diarrhea and hypokalemia. No cause was found. In March 1981 a routine electrolyte screening was done before a dental surgical procedure. Initial evaluation showed hypokalemia, thought to be due to Bartter's syndrome,[1] and she was referred to our clinic. She had moderate muscle weakness and said she had not eaten any licorice, laxatives or diuretics. Findings on physical examination were unremarkable and there was no evidence of postural blood pressure variation or edema.

Laboratory results included sodium 139, potassium 2.8, chloride 86 and bicarbonate 36.3 mEq per liter; BUN 17 mg per dl; a supine plasma renin activity determination measured 12.7 ng per ml per hour and serum aldosterone 20 ng per dl. Spot urine analysis showed a sodium value of 176 and a potassium level of 61 mEq per liter. The urine electrolyte loss suggested the possibility of diuretic use, which was confirmed by the finding of urinary furosemide concentration of 7 μg per ml. Tests of urine were negative for thiazide concentrations.

She was confronted but would not admit to furosemide abuse. She asked that we repeat the urine tests. Repeat screening of the urine showed it to be negative for furosemide, but positive for thiazide (22 μg per ml). She continued to deny intermittent use of different diuretics and she was referred back to her primary physician for further therapy.

Methods

Electrolyte determinations were done on an autoanalyzer (Technicon). Analysis of plasma renin activity was done by University of California laboratories.[2] Serum aldosterone radioimmunoassays were carried out at Nichols Institute, San Pedro, California.[3] Both urine furosemide and urine thiazide quantities were determined by Hines Laboratory, San Francisco. Urine furosemide concentrations were measured using a fluorometric assay.[4] Values less than 2 μg per ml may be related to nonspecific urinary fluorescent activity. Values greater than 2 μg per ml are specific. Urine thiazide determinations were done colorimetrically.[5] The assay is specific for thiazides but cannot differentiate chlorothiazide from hydrochlorothiazide.

Discussion

The known ingestion of furosemide and thiazide diuretics is associated with multiple and easily recognizable side effects.[6,7] When diuretic use is covert, however, the consequences may be ascribed to myriad other causes.

Diuretic abuse is most common in women who may be health professionals and tend to be preoccupied with either weight loss or edema.[8-10] They may go to incredible lengths to conceal their medication habit. Patients have undergone selective venous catheterization,[11] placement of a shunt permitting venous access for prolonged potassium administration[11] and renal biopsy[12,13] before admitting to taking a diuretic.

Electrolyte abnormalities are the most common side effects of diuretic use[6] and abuse.[11-21] Hyponatremia, as seen in case 1, occurs when impaired free water clearance caused by diuretics is accompanied by increased water ingestion.[22] Hypokalemia and hypochloremic alkalosis can be consequences of both thiazide and furosemide use.[23,24] The hypochloremic acidosis of case 1 suggests that thiazides alone could not have induced the electrolyte acid-base abnormality. The low serum bicarbonate level is probably due to diarrhea, and the hypochloremia to ingestion of diuretics. A mixed gastrointestinal-diuretic cause is further supported by the low urine sodium and positive result of urine thiazide assay.

Both furosemide and thiazides can produce volume depletion. The patient in case 1 had sufficient volume contraction to cause mild postural hypotension, xerostomia and an elevated BUN level. Given her denial of gastrointestinal symptoms or diuretic use, studies had been done for Sjögren's syndrome and renal disease.

Volume depletion results in increased renin production by the juxtaglomerular apparatus, with secondary rise in aldosterone levels.[25] Both furosemide and thiazides can induce hypokalemia by direct renal tubular effects.[23] The hyperaldosteronism due to volume depletion further contributes to renal potassium wasting. The consequent hypokalemia can present as muscle weakness or may be interpreted as arthralgias (case 1). The combination of hypokalemic-hypochloremic alkalosis and increased serum renin and aldosterone concentrations that is seen in association with diuretic abuse can mimic Bartter's syndrome.[12-16,26,27]

In 1962 Bartter and co-workers[1] described a syndrome of short stature, hypokalemia, increased renin and aldosterone levels, and normal blood pressure. Juxtaglomerular apparatus hyperplasia was found on examination of a renal biopsy specimen and there was a decreased hypertensive response to angiotensin infusion. Recent studies have helped clarify the pathophysiology of Bartter's syndrome. Although not firmly established, the primary defect would appear to be decreased chloride reabsorption in the ascending limb of the loop of Henle.[28] This results in increased urinary chloride and potassium excretion and in hypokalemia. The increased renal prostaglandin production that occurs in Bartter's syndrome[29] may be due to hypokalemia,[30] but other stimuli such as angiotensin II, bradykinin, vasopressin and polyuria have been suggested.[31] Prostaglandins increase renin production by the juxtaglomerular cells. Constant juxtaglomerular apparatus stimulation results in juxtaglomerular cell hyperplasia. Renin via angiotensin II augments adrenal production of aldosterone. Prostaglandins may also increase synthesis of bradykinin, which, in conjunction with volume depletion, prevents hypertension in the face of endogenous or exogenous angiotensin II.[32,33]

The primary action of furosemide is to inhibit chloride reabsorption in the ascending limb of Henle's loop.[34] Thiazides also promote urinary excretion of chloride.[35] If the chloride reabsorption defect proposed for Bartter's syndrome is correct, it follows that furosemide and thiazides could induce a state very analogous to Bartter's syndrome (pseudo-Bartter's syndrome, type I). In fact, examination of renal biopsy specimens in two cases of covert furosemide use have confirmed juxtaglomerular cell hyperplasia.[12,13] The only way to differentiate true Bartter's syndrome from diuretic-induced pseudo-Bartter's syndrome, type I, would seem to be by repeatedly screening for furosemide and thiazides.

Prolonged laxative abuse[18,36,37] or emesis,[38,39] or both, can also produce a pseudo-Bartter's syndrome, accompanied by juxtaglomerular apparatus hyperplasia.[36,37,39] In contrast with true Bartter's syndrome or the diuretic-induced states, gastrointestinal electrolyte loss is accompanied by low urine chloride levels[38] (pseudo-Bartter's, type II). Combined diuretic and laxative abuse with variable urinary electrolyte measurements has been described.[18]

Pseudo-Bartter's syndrome may account for many of the cases presenting in adulthood. The true syndrome is usually diagnosed in children[40] but has been reported in adults as old as age 74.[41] Some of the adult patients have had symptoms dating to early childhood, and in others surreptitious vomiting or laxative or diuretic abuse had not been eliminated. Therefore, true adult Bartter's syndrome would seem to be a very rare occurrence. Repeated determinations of urinary chloride and diuretic concentrations should be done before embarking on costly and invasive investigations for the adult syndrome.

Thiazides inhibit urine calcium excretion[42] and can raise both serum total[42] and ionized[43] calcium levels. An association between thiazide use and hyperparathyroidism has been suggested.[44] The hypercalcemia in case 1 was probably due to volume depletion, as indicated by the high serum albumin and normal ionized calcium levels. Nevertheless, the persistently mild hypercalcemia had prompted investigations for sarcoidosis and hyperparathyroidism.

Thiazide use probably contributed to the repeated bouts of pancreatitis seen in case 1. An association of thiazides and pancreatitis is described.[45,46] Cholelithiasis, which was not present in our patient, may also be thiazide-related.[47]

Other side effects of thiazide or furosemide use might occur with covert ingestion (Table 1). These include cardiac arrhythmias,[24] thrombocytopenia,[48] skin rash,[49,50] hypomagnesemia with or without hypocalcemia,[51] auditory abnormalities,[52] hyperuricemia,[53] carbohydrate intolerance,[54] interstitial nephritis[55] and cyclic edema.[8]

The patient in case 2 had a history of edema. MacGregor and colleagues[8,56] have studied patients with idiopathic edema. All were taking diuretics. Several patients had the onset of swelling only after beginning diuretic use. Most had worsening of edema after they stopped taking these drugs, with resolution within two to three weeks. High renin and aldosterone concentrations were found in these patients for up to four weeks after diuretic intake was stopped. Thus, many cases of idiopathic edema may be due to overt or covert diuretic ingestion.

TABLE 1.—*Clinical Abnormalities That Suggest Covert Diuretic Use*

Taking Diuretics Only
Electrolyte abnormalities
Hyponatremia
Hypokalemia
Hypochloremic alkalosis
Hypercalcemia, hypomagnesemia, hypocalcemia
Other abnormalities
Polyuria, nocturia
Volume depletion, xerostomia
Pancreatitis
Skin rash
Thrombocytopenia
Hearing loss
Cardiac arrhythmias
Elevated blood urea nitrogen and/or interstitial nephritis
Hyperuricemia
Hyperglycemia
Taking Diuretics Plus Laxatives
Hypokalemic acidosis
All of the above listed abnormalities

Summary

Covert diuretic abuse can present in a variety of diagnostic disguises. The general availability of assays for thiazides and furosemide can help prevent expensive and potentially dangerous investigations. Use of these assays is suggested for patients with electrolyte acid-base disturbances of obscure origin. Diuretic screening also seems indicated when prototype patients (that is, a woman, a health worker and so forth) have disorders of unknown cause accompanied by electrolyte abnormalities and the clinical features can be associated with known diuretic toxicity.

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Isolated Cortical Blindness in Pregnancy

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ISOLATED CORTICAL BLINDNESS due to preeclampsia or eclampsia is unusual. Recently we saw two patients with this syndrome. Reports of their cases

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No reprints are available.